Routes of Transmission and the Introduction of Foreign Animal Diseases

overview 2
part 3

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Examples of Introductions by Different Routes of Transmission

Now that you have reviewed the introductory material, we are going to investigate actual modes of introduction of exotic diseases. Exotic diseases can be introduced into a country by the movement of fomites, vectors, infected animals, animal products, or by the emergence of new diseases or new variants. The likelihood of these introduction events can be decreased through strict surveillance, precautionary practices, and improved biosecurity.

In order to enhance your understanding of the modes of introduction, we will present examples of each of these potential routes for you to review. As you work through the examples, you may wish to review the definitions and examples presented in the first half of this chapter or explore the expanded information on specific diseases provided in the appendix of diseases in the back of this book.

Fomites and the Introduction of Foot and Mouth Disease

How could these filthy boots cause ulcers on the hooves of a calf?

By serving as a fomite that carries a pathogen from one animal to another. As you may recall, a fomite is an inanimate object on which a pathogen can be conveyed. Disease transmission occurs via shared physical contact between the object and the animal. Classic examples of fomites are contaminated footwear, veterinary equipment, needles, clothing, eating or drinking containers, cages, bedding, dander, restraint devices, and transportation vehicles. The control of fomites can be vital in preventing the spread of some diseases, such as foot and mouth disease (FMD).

In 2001, the United Kingdom was facing a crisis of epic proportions. In February of that year, a veterinary inspector found lesions suggestive of FMD on several pigs in an abattoir. The movement of animals in the U.K. was halted and the pigs were traced back to an infected farm in Northumberland. But by this time the FMD virus had spread, resulting in an epidemic that devastated the livestock industry and economy of the U.K. Before the outbreak was finally brought under control, more than 2,000 cases of FMD had been diagnosed, more than four million animals had been slaughtered, and foci of infection had spread to France, Ireland, and the Netherlands. FMD is currently endemic in parts of Asia, Africa, the Middle East, and South America. Although the U.K. and many other countries have eradicated this disease within their borders, re–introduction of the virus can lead to massive epidemics such as the one seen in 2001. How is this disease introduced and spread to new areas?

One method of spread is by fomites. Under the right conditions, the foot and mouth disease virus can persist for days to weeks in the environment. Virus particles that contaminate footwear, clothing, transportation equipment, and other fomites can be transported long distances. Although FMD is not infectious to humans, humans can carry the virus in their nasal passages for one to two days, and may transport the virus on clothing. Because of the high risk of transmission by humans and fomites, officials target people to try to prevent the introduction of the virus. Travelers who have visited FMD–positive countries and who have been on farms or contacted farm animals are required to declare this information at customs. Officials inspect their baggage and disinfect soiled footwear with bleach and detergents. Infectious virus can also be found in animal products such as meat–in–bone, milk, bones, glands, and cheese. All ruminant and swine products are confiscated and travelers are asked to wash their clothing prior to returning home to an FMD–free area. In addition, travelers are instructed to stay off all farms for at least five days after their return .

Vectors and the Introduction of West Nile Virus

How could this insect cause circling, convulsions and ataxia in horses?

By serving as a vector for a pathogen that causes encephalitis. Vectors may be either biological vectors that are persistently infected and allow the pathogen to develop and reproduce or mechanical vectors on which the pathogen resides for a short period of time. Because they are persistently infected, biological vectors are more likely to introduce exotic disease agents to new areas than are mechanical vectors. The entry of the West Nile virus (WNV) into the United States in 1999 may have been an example of a vector–borne introduction. West Nile virus causes encephalitis in humans, horses, and some species of birds. Mosquito vectors spread this virus; in the U.S., members of both *Culex* and *Aedes* are capable of transmitting it. Until recently, West Nile encephalitis was found only in Africa, Europe, the Middle East, west and central Asia, and Oceania. In the summer and fall of 1999, West Nile virus was identified in dead birds in and around the Bronx zoo and, concurrently, in several cases of human and equine encephalitis in New York. How did West Nile virus enter the United States?

Scientists with the Centers for Disease Control and Prevention suspect that West Nile virus was present in the U.S. by at least early summer of 1999. The virus' point of origination is unclear but isolates from the U.S. most closely resemble virus strains from Israel. A possible scenario for introduction is that an infected mosquito vector from an endemic area traveled to North America on an airplane and was inadvertently released from the airplane, resulting in the introduction of the virus into native bird populations

Various species of birds can serve as reservoirs for West Nile virus. To date, over 110 species of birds, predominately corvids (crows, ravens and their relatives), have tested positive for the virus. Birds with severe infections suffer high morbidity and mortality rates, but typically develop life–long immunity after exposure and a short viremia. Mosquitoes acquire the virus when they feed on the infected reservoir birds. Many mammals, including humans and horses, are incidental hosts that become infected when fed upon by an infected mosquito. Although mammals do not develop levels of viremia sufficient to infect mosquitoes, and thus cannot serve as reservoirs, infections in mammals may result in severe, potentially fatal meningoencephalitis. Some adult *Culex* species in the northeastern U.S. survive the winter and thus are able to overwinter the virus. Either these mosquitoes or infected birds may have been responsible for the persistence of the virus and the re–emergence of West Nile encephalitis in the summers of 2000, 2001, and 2002. In spite of eradication efforts targeted at the mosquito vector, West Nile virus has now spread throughout much of the U.S.

Infected Animals and the Introduction of *Elaphostrongylus rangiferi*

How could this apparently healthy animal cause neurologic disease in caribou herds throughout Newfoundland?

By carrying an exotic disease agent that can become established in animal herds, the environment, or vectors native to the area. When it enters a country, a healthy–looking animal may be incubating an exotic disease or serving as an asymptomatic reservoir for a pathogen. Naïve animals can become infected by direct or indirect contact with the animal. Introduction of infected animals is thought to have been responsible for bringing cerebrospinal elaphostrongylosis to native Canadian caribou.

Cerebrospinal elaphostrongylosis is a severe neurological disease of cervids, sheep, and goats caused by the nematode *Elaphostrongylus rangiferi*. *E. rangiferi* has an indirect life cycle. Caribou or reindeer are the definitive hosts and shed larvae in their feces. The larvae penetrate the footpad of a snail or slug intermediate host to develop into the infective stage. Caribou or reindeer then ingest infected snails or slugs while feeding. Once in the host, the parasites migrate through the spinal cord and brain before reaching the muscles of the shoulder and hindlimbs where they mature. Eggs passed by adults are carried via the bloodstream to the lungs where they hatch into larvae, cross the alveoli, travel up the respiratory tree, are swallowed, and are then excreted in the feces.

Cerebral elaphostrongylosis was first recognized in Newfoundland in the 1970s. It was introduced to Newfoundland in the early 1900s when infected reindeer were imported from Norway to establish herds for food and draft animals. Once in Newfoundland, the reindeer traveled across native caribou range and some escaped, most likely commingling with caribou. At present, most of the caribou herds in Newfoundland are infected with *E. rangiferi* although mainland herds appear to remain free of infection. To prevent introductions such as this, animals entering a country are now quarantined and tested for exotic diseases before they are allowed to mingle with native populations.

Animal Products and the Introduction of African Swine Fever

How could this ham sandwich cause fever, anorexia, and reddened skin in pigs?

By carrying a pathogen that can survive for a period of time in animal products. Animal products can transmit disease agents when they are discarded near farms or are deliberately fed to susceptible animals. The practice of feeding uncooked scraps to pigs, now discouraged in many countries, is a typical route of introduction for several exotic swine diseases, including African swine fever (ASF). To prevent the introduction of African swine fever, officials tightly regulate the movement of pork products from endemic to ASF–free countries.

The African swine fever virus is a DNA virus that is newly classified in its own group, Asfarviridae but has similarities to both Iridovirus and Poxvirus. Susceptible animals include domestic and feral pigs, wart hogs, and peccaries. African swine fever is currently endemic in Sub–Saharan Africa and has been present but is now eradicated from Cuba, Haiti, and the Dominican Republic. The disease is characterized by fever, depression, occasional hemorrhagic disorders, and sometimes death. Transmission occurs via direct contact with infected animals or indirect contact via ticks, fomites, or feeding infected meat to pigs.

Preventing the introduction of African swine fever requires strict regulation of the importation of pigs and pork products. The virus is very hardy and can survive for months within pork products or in infected ticks. Importation of pork products from an infected herd poses the greatest risk for introduction of the disease. Official regulations control the importation of animal products from countries known to harbor OIE–regulated diseases. In addition, animal products are confiscated from travelers returning to the United States.

The Emergence of New Diseases and the Development of Bovine Spongiform Encephalopathy

How could this animal develop an exotic disease without exposure to any known pathogen?

By contracting a previously unknown animal disease. Each year new strains of pathogens are discovered, and occasionally completely novel pathogens are recognized. The transmissible spongiform encephalopathies (TSEs) are examples of a novel pathogen type that has emerged in several forms over the last few decades. Transmissible spongiform encephalopathies are fatal neurologic diseases found in a number of species including sheep, goats, cattle, elk, deer, exotic ruminants, cats, mink, and humans. Animals with TSEs develop progressive neurologic degeneration and exhibit incoordination, ataxia, nervousness or aggression, and decreased production despite continued appetite. The agents of TSEs have not been completely characterized, but the pathogen appears to be smaller than a virus. Three major theories have been put forth to describe the causative agents of TSEs: (1) the agent is a prion with an exclusively host–coded protein that is modified to a partially protease–resistant form after infection, (2) the agent is a virus with unusual characteristics, or (3) the agent is a small, noncoding regulatory nucleic acid coated with a host–derived protective protein. In most cases, TSE agents seem to be spread orally.

Some TSEs, including scrapie in sheep and chronic wasting disease in deer and elk, have been recognized for a number of years. Others appear to be new variants. Bovine spongiform encephalopathy (BSE), commonly referred to as Mad Cow Disease, was first diagnosed in Great Britain in 1986. By the time BSE was recognized, a full–blown epidemic had begun. In spite of control measures, the number of cases escalated each year, finally peaking in 1993. Epidemiological evidence suggests that BSE developed in British cattle when they consumed feed that used contaminated meat–and–bone meal as a protein source. The relationship of BSE to other TSEs is not fully understood, but cases of new variant Creutzfeldt Jakob disease (vCJD), a human TSE, have been causally linked to exposure to BSE. In the UK, the use of mammalian meat–and–bone meal in feed for all food–producing animals is now prohibited; in addition, because older animals are more likely to be infected, carcasses from animals more than 30 months of age are no longer allowed to be used as domestic animal or human food.